1. A method of treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient the method comprising administering to the patient an effective amount of a compound that is able to reduce the production, absorption and/or the effect of an endotoxin (lipopolysaccharide; LPS).

2. (amended) The method of claim 1, wherein the heart failure includes endotoxin-mediated immune activation.

3. (amended) A method according to claim 1 wherein the compound is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule.

4. (amended) A method according to claim 1 wherein, the compound is able to reduce the available endotoxin in the patient.

5. (amended) A method according to claim 1 wherein the compound is a bile acid.

6. (amended) A method according to claim 1 wherein the bile acid is any one of ursodesoxycholic acid, chemodeoxycholic acid, dehydrocholic acid, cholic acid and deoxycholic acid.

7. (amended) A method according to claim 1 wherein the compound is LPS binding protein, bactericidal/permeability increasing protein (BPI), a lipoprotein, for instance but not exclusively low density lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL),

apolipoprotein (a), a lipoprotein mixture or an antibody capable of binding to endotoxin (lipopolysaccharide; LPS).

8. (amended) A method according to claim 1 wherein the compound is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut.

9.(amended) A method according to claim 1 wherein the compound is able to reduce the absorption of endotoxin by the patient from the gut.

10.(amended) A method according to claim 1 wherein the compound is able to substantially reduce tile availability of endotoxin (lipopolysaccharide) for absorption from the gut, such that the amount of endotoxin that is absorbed is reduced or is less biologically active.

11.(amended) A method according to claim 1 wherein the compound is activated charcoal activated carbon, Fuller's earth, attapulgite, kaolin, bentonite or a clay or colostrum of human, bovine, or other mamallian origin

12.(amended) A method according to claim 1, wherein the compound is an antibacterial agent.

13.(amended) A method according to claim 12 wherein the antibacterial agent is active in the gut.

14.(amended) A method according of claim 12 wherein the antibacterial agent is able to substantially reduce the amount of bacteria and/or free endotoxin (lipopolysaccharide) in the gut.

15.(amended) A method according of claim 12 wherein the antibacterial agent is largely unabsorbed from the gut.

16. (amended) A method according of claim 12 wherein the antibacterial agent is an antibiotic, for instance but not exclusively non-absorbable antibiotics like neomycin, tobramycin, ampbotericin B, and colistin.

17.(amended) A method according to claim 1 wherein the compound is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS).

18. (amended) A method according to claim 17 wherein the compound is able to decrease the cytokine production by a cell in response to endotoxin (lipopolysaccharide; LPS).

19. (amended) A method according to claim 17 wherein the compound is an antibody able to bind the CD14 receptor, soluble

CD14 receptor or an antibody or non-functional agonist of a toll-like receptor.

20.(amended) A method according to claim 17 wherein the compound is able to inhibit signalling *via* the CD14 receptor or via a toll-like receptor.

21. (amended) A method according to claim 1 wherein the compound is able to reduce the permeability of the gut wall to bacteria and/or endotoxin (lipopolysaccharide; LPS).

22.(amended) A method according to claim 1 wherein the agent is able to reduce the amount of bacteria and/or free endotoxin (lipopolysaccharide) that is able to translocate from the gut into the circulation of the patient.

23.(amended) A method according to claim 21, 22 wherein the agent is largely unabsorbed from the gut.

24.(amended) A method according to claim 1 wherein the agent is IGF-1, allopurinol, oxipurinol, or any other unspecific xanthine oxidase inhibitor, or a specific xanthine oxidase inhibitor (like TMX-67), liquorice or its

derivatives, carbenoxolone, an alginate, sulfacrate or an agent that may form a hydrogel.

25.(amended) A method according to claim 1 wherein the compound is administered orally.

26.(amended) A method according to claim 1 wherein the compound is administered intravenously.

27.(amended) A method according to claim 1 wherein the compound is administered rectally.

43. (amended) The method claim 1 wherein a HMG-coenzyme A-reductase inhibitor that is able to increase lipoprotein levels and is not used to lower LDL / cholesterol levels is administered to the patient.

45.(amended) The method claim 1 wherein a diuretic is administered to the patient.

46.(amended) A pharmaceutical formulation according to claim 78, wherein the compound is comprising bile acid or BPI

or LPS binding protein, a lipoprotein, a lipoprotein mixture, or an antibody capable of binding LPS.

47. (amended) The pharmaceutical formulation according to claim 78 comprising a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut and a diuretic.

48. (amended) The pharmaceutical formulation according to claim 78 comprising an antibacterial agent and a diuretic.

49. (amended) The pharmaceutical formulation according to claim 78 comprising a compound that is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS) and a diuretic.

50. (amended) The pharmaceutical formulation according to claim 78 comprising an agent that is able to reduce the permeability of the gut wall to bacteria and/or endotoxin (LPS) and a diuretic.

53. A method of treating or ameliorating body wasting or cachexia in a patient with liver cirrhosis, chronic

obstructive pulmonary disease, chronic renal failure, diabetes, rheumatoid arthritis in a patient the method comprising administering to the patient an effective amount of a compound that is able to reduce the production, absorption and/or the effect of an 10 endotoxin (lipopolysaccharide; LPS).

54. (amended) The method of claim 53, wherein the patient's condition further comprises endotoxin-mediated immune activation.

55. (amended) A method according to claim 53 wherein the compound is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule.

56. (amended) A method according to claim 53 wherein the compound is able to reduce the available endotoxin in the patient.

57. (amended) A method according to claim 53 wherein the compound is a bile acid.

58. (amended) A method according to claim 57 wherein the bile acid is any one of ursodesoxycholic acid,

chemodeoxycholic acid, dehydrocholic acid, cholic acid and deoxycholic acid.

59. (amended) A method according to claim 53 wherein the compound is LPS binding protein.

60. (amended) A method according to claim 53 wherein the compound is bactericidal/permeability increasing protein (BPI).

61. (amended) A method according to claim 53 wherein the compound is a lipoprotein.

62. (amended) A method according to claim 53 wherein the compound is a combination of LPS binding protein and a lipoprotein.

63. (amended) A method according to claim 53 wherein the compound is an antibody capable of binding to endotoxin (lipopolysaccharide; LPS).

65. (amended) A method according to claim 53 wherein the compound is an antibody able to bind to the CD 14 receptor.

66. (amended) A method according to claim 53 wherein the compound is a soluble CD14 receptor.

67. (amended) A method according to claim 53 wherein the compound is a drug blocking effectively signaling through toll-like receptors.

68. (amended) A method according to claim 53 wherein the compound is colostrum of human, bovine, or other mamallian origin.

69. (amended) A method according to claim 53 wherein the compound is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS).

70. (amended) A method according to claim 53 wherein the compound is able to decrease the cytokine production by a cell in response to endotoxin (lipopolysaccharide; LPS).

72. (amended) A method according to claim 53 wherein the compound is administered orally.

A6

73. (amended) A method according to claim 53 wherein the compound is administered intravenously.

74. A method according to claim 53 wherein the compound is administered rectally.

A7

76. (new) A method according to claim 17, wherein the compound is an antibody able to bind the CD14 receptor, soluble CD14 receptor or an antibody or non-functional agonist against the toll-like receptor 4 and 2.

77. (new) A method according to claim 17, wherein the compound is able to inhibit signalling *via* the CD14 receptor or via the toll-like receptor 4 and 2.

78. (new) A pharmaceutical formulation comprising a diuretic and a compound chosen from the group consisting of:

a) bile acid or BI or LPS binding protein, a lipoprotein, a lipoprotein mixture, or an antibody capable of binding LPS;

b) a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut;

c) an antibacterial agent;

d) a compound that is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS); and

e) an agent that is able to reduce the premeability of the put wall to bacteria and/or endotoxin (LPS).

79. (new) A pharmaceutical formulation according to claim 78, wherein the compound is a lipoprotein chosen from the group consisting of low density lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL), and apolipoprotein (a).

80. (new) A method according to claim 53, wherein the compound is a lipoprotein chosen from the group consisting of low density lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL), apolipoprotein (a), a lipoprotein mixture.

81. (new) A method according to claim 53, wherein the compound is a drug blocking effectively signaling through the toll-like receptor 4 and toll-like receptor 2.